

L20 ANSWER 1 OF 2 MEDLINE on STN
 AN 2002430653 MEDLINE
 DN PubMed ID: 12187043
 TI Subsequent activation of mitogen-activated protein kinase after adhesion of transitional cell cancer cells to fibronectin.
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 SO Urologia internationalis, (2002) Vol. 69, No. 2, pp. 125-8. Journal code: 0417373. ISSN: 0042-1138.
 CY Switzerland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 200210
 ED Entered STN: 21 Aug 2002
 Last Updated on STN: 29 Oct 2002
 Entered Medline: 28 Oct 2002
 AB INTRODUCTION: In the process of tumor invasion and metastasis, interactions between tumor cells and extracellular matrix play a crucial role. Recently, it was shown that fibronectin binding to fibronectin receptor promotes mitogen-activated protein kinase (MAPK) activation after tyrosine phosphorylation of focal adhesion kinase (FAK). We investigated these signal transduction events in transitional cell cancer (TCC) cells. MATERIALS AND METHODS: (1) The adhesion of T24 cells, a fibronectin-receptor-positive TCC cell line, to fibronectin was investigated; (2) the MAPK activation after fibronectin stimulation in bladder cancer cell lines was examined by Western blotting using an antiactive MAPK antibody, and (3) FAK, Sos, and Grb-2 were also examined by Western blot analysis. RESULTS AND CONCLUSIONS: T24 cells adhered to fibronectin-coated dishes more quickly than to the noncoated dishes. Fibronectin stimulation induced activation of MAPK in T24, SCaBER, and HT1376 cells. However, activated MAPK was not detected in RT4 cells which do not express alpha(5)beta(1) integrin (major fibronectin receptor) after fibronectin stimulation. T24, SCaBER, and HT1376 expressed FAK and Sos. RT4 showed little FAK and Sos expression. Grb-2 was expressed in all cell lines. Adhesion of fibronectin-receptor-positive TCC cells to fibronectin activates the MAPK cascade, possibly resulting in activation of tumor cells.
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L17 ANSWER 7 OF 27 MEDLINE on STN
AN 2004528586 MEDLINE
DN PubMed ID: 15498297
TI The heat shock protein 70 antisense
oligomers enhance the sensitivity of bladder cancer
cell EJ to mitomycin C.
AU He Ling-feng; Hou Shu-kun; Yan Zheng; Ren Liang; Wang Shen-wu
CS Department of Urology, Peking University People's Hospital, Beijing
100044, China.
SO Zhonghua wai ke za zhi [Chinese journal of surgery], (2004 Sep 22) Vol.
42, No. 18, pp. 1108-10.
Journal code: 0153611. ISSN: 0529-5815.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA Chinese
FS Priority Journals
EM 200609
ED Entered STN: 23 Oct 2004
Last Updated on STN: 11 Feb 2005
Entered Medline: 22 Sep 2006
AB OBJECTIVE: To investigate whether the heat shock
protein (HSP) 70 antisense oligomers can enhance the
sensitivity of bladder cancer cell EJ to mitomycin C.
METHODS: The HSP70 mRNA of EJ cells was blocked by the 10
micromol/L HSP70 antisense oligomers, while its effect on cell
growth was evaluated by methyl thiazolyl tetrazolium (MTT) and colony
forming ability test. RESULTS: The HSP70 expressions in
HSP70 antisense treated group were lower than the corresponding
sense and nonsense treated groups ($P < 0.01$). While, the increased
sensitivity of EJ to mitomycin C was found in antisense treated group,
compared with the corresponding sense and nonsense treated groups ($P < 0.01$). CONCLUSION: The sensitivity of bladder cancer
cell EJ to mitomycin C was enhanced by the blockage of the HSP70
expression.

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